

Refer to: Cello JP, Crass RA, Federle MP: Colonic varices: An unusual source of lower gastrointestinal hemorrhage. *West J Med* 136:251-255, Mar 1982

Colonic Varices: An Unusual Source of Lower Gastrointestinal Hemorrhage

JOHN P. CELLO, MD
RICHARD A. CRASS, MD
MICHAEL P. FEDERLE, MD
San Francisco

PORTAL VENOUS HYPERTENSION from hepatic cirrhosis commonly leads to a dilation of submucosal venous channels in regions of the gastrointestinal tract where splanchnic and systemic veins anastomose.¹ Esophageal varices are clinically the most important and the most frequent portasystemic venous shunts occurring in patients with portal hypertension, these varices being frequently associated with spontaneous massive upper gastrointestinal hemorrhage. Rectal varices, mesenteric serosal varices, dilated paraumbilical veins (caput medusae) and spontaneous retroperitoneal spleno-renal venous shunts likewise may develop in patients with substantial portal hypertension, but they are rarely associated with significant hemorrhage. Other regions of the gastrointestinal tract and abdomen, though subjected to the same high venous pressures, are rarely involved with significant hemorrhage from portal hypertension. We present the case of a patient with Laennec's cirrhosis in whom severe colonic hemorrhage developed from right colonic varices. This case illustrates the necessity of considering this entity in explaining substantial lower gastrointestinal hemorrhage in cirrhotic patients.

Report of a Case

A 35-year-old woman with a long history of alcohol abuse and Laennec's cirrhosis proved on biopsy was admitted with cramping right lower

quadrant abdominal discomfort and the passage of multiple bright red bloody stools. Several years before admission she had an exploratory laparotomy; no appendectomy or bowel resection was done at that time. Four months before the present hospital admission she was admitted to another hospital with melena and rectal hemorrhage of bright red blood. Endoscopy at that time showed large nonbleeding esophageal varices and an active ulcer of the duodenal bulb. She was treated with antacids for peptic ulcer disease and did well over the next four months. She continued to consume up to a pint of alcohol a day, but stated that she did not have epigastric pain, melena, hematemesis or melanemesis during the four months before admission.

On the morning of admission she felt well except for mild cramping and lower abdominal discomfort and she spontaneously passed a large stool of clotted bright red blood. She then felt lightheaded and diaphoretic and vomited her breakfast. She stated she has never had hemorrhoids, colitis, altered bowel habits, diverticular disease or unknown bleeding disorders. On physical examination she was noted to be well developed, well nourished and somewhat older in appearance than her stated age. Blood pressure was 90/60 mm of mercury and pulse 124 beats per minute. Examination of the chest and heart showed no abnormalities. Her abdomen was nontender but did have a fluid wave and shifting dullness to percussion suggestive of ascites. The liver was palpable just beneath the right costal margin and had a 14-cm span to percussion. The splenic tip was also palpable. On rectal examination no obvious external hemorrhoids or palpable anorectal masses were found but there was bright red blood on the examining glove.

A sigmoidoscopy done to 20 cm showed fresh blood overlying normal mucosa without ulceration, mass lesions or rectal varices. There were no internal or external hemorrhoids. A nasogastric tube was passed soon after admission and returned clear yellow bilious fluid. Guaiac test of the nasogastric aspirate was negative. Her admission laboratory studies showed a hematocrit of 20 percent, a prothrombin time of 12.2 seconds (control 11.0 seconds), a partial thromboplastin time of 32.9 seconds, normal serum electrolytes, blood urea nitrogen and serum creatinine. Other laboratory test results included the following: serum albumin 2.7 grams per dl, serum total protein 5 grams per dl, serum total bilirubin 1.4

From the Departments of Medicine (JPC), Surgery (RAC) and Radiology (MPF), San Francisco General Hospital and the University of California, San Francisco.

Submitted, revised, May 26, 1981.

Reprint requests to: John P. Cello, MD, San Francisco General Hospital, 5H-9, San Francisco, CA 94110.

CASE REPORTS

mg per dl, serum aspartate aminotransferase (formerly serum glutamic oxaloacetic transaminase, SGOT) 37 IU per liter and alkaline phosphatase 81 IU per liter (normal <80). X-ray studies of the chest and plain films of the abdomen were normal.

Initially a transfusion with five units of packed red cells was carried out to maintain a hematocrit in the 30 percent range. Continued intermittent bright and dark red blood clots were passed through the rectum. At upper gastrointestinal endoscopy there were large esophageal varices extending into the cardia of the stomach. The gastric and duodenal mucosa was completely normal. Sequential scintigraphy with sodium pertechnetate Tc 99m and technetium Tc 99m sulfur colloid failed to show either ectopic gastric mucosa of a Meckel's diverticulum or extravasation of radionuclide into the bowel.

The patient's colon was lavaged clear by saline enemas and on the first hospital day a colonoscopy was done to the level of the midtransverse colon.

The colonic mucosa was completely normal to the proximal transverse colon without evidence of diverticula, vascular malformations, dilated submucosal veins or mucosal erosions. Bright red blood was seen coming from the hepatic flexure into the transverse colon. Because of the large amount of blood coming from the hepatic flexure, the examination could not be extended to the ascending colon and cecum.

On the morning of the second hospital day, the patient underwent abdominal angiography while still hemorrhaging. The inferior and superior mesenteric arteries and celiac artery were selectively injected following transcatheter injection of 25 mg of tolazoline hydrochloride (Priscoline hydrochloride) to enhance the mesenteric venous views. Filming was carried out through the venous phase. No intraluminal extravasation of contrast material was shown, despite continued active hemorrhage requiring multiple blood transfusions during the study. Large dilated cecal and ascending colonic veins draining into a large right ovarian

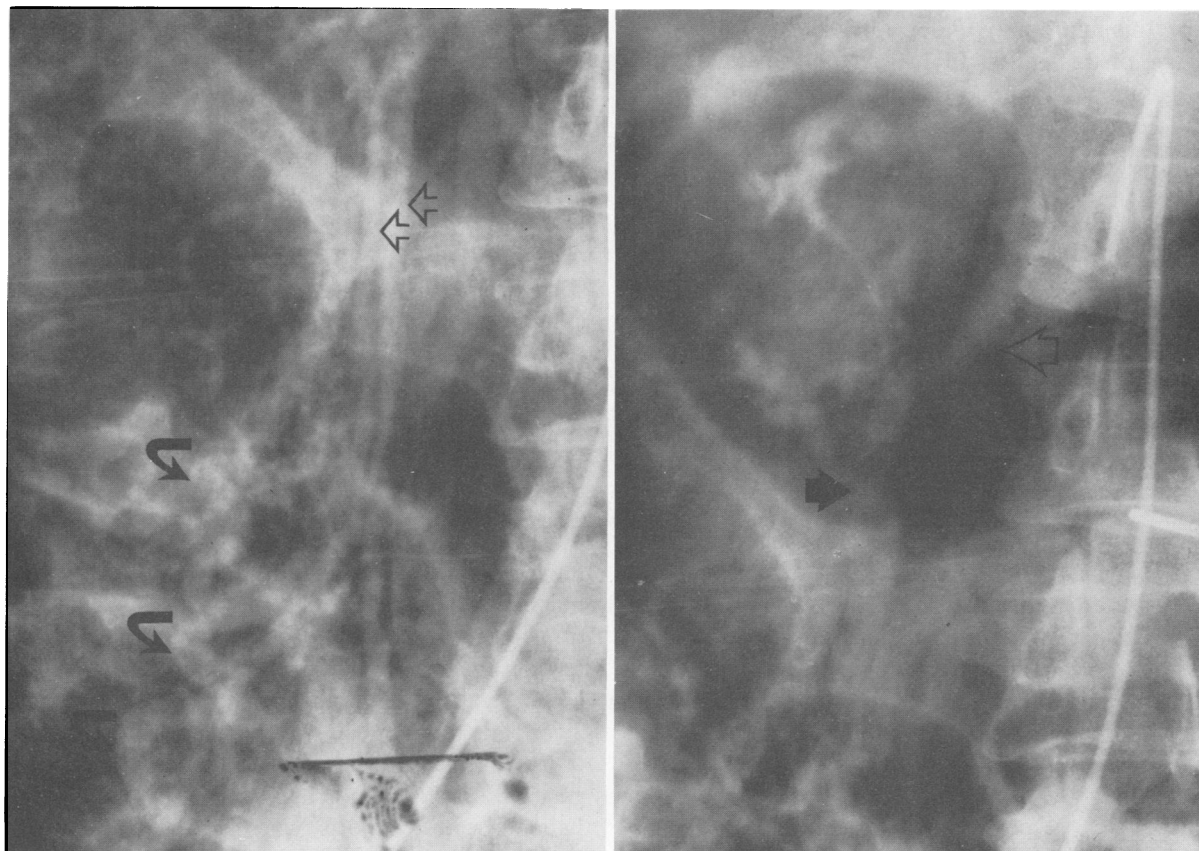


Figure 1.—Colonic varices. **Left**, early venous phase of superior mesenteric angiogram. Dilated tortuous veins (varices) (curved arrows) opacified in area of cecum. Double right ureter (open arrows) incidentally noted. **Right**, late venous phase. Dilated right ovarian vein (solid arrow) drains varices. The superior mesenteric vein (open arrow) is also opacified.

vein were identified, however (Figure 1). No other mesenteric varices were seen.

Active hemorrhage continued despite intravenously administered vasopressin, 0.4 units a minute. She required 12 units of packed red cells during the first 36 hours of hospital admission. At laparotomy, 48 hours after admission, a large cirrhotic liver was noted; a portal venous pressure of 46 cm of saline was measured (normal <20 cm of saline). An end-to-side portacaval shunt was made. Following the shunt procedure, the portal pressure was 21 cm of saline. The patient ceased hemorrhaging following portacaval shunting and had an uneventful postoperative course. She was discharged on the 14th postoperative day and remains well six months after surgery, without further rectal bleeding, anemia or guaiac-positive stool.

Discussion

Although fewer than 50 cases of mesenteric variceal hemorrhage mainly involving the colon have been reported in the English-language literature, this figure may underrepresent the true prevalence of this entity. In 110 cirrhotic patients undergoing percutaneous transhepatic portography, three demonstrated superior mesenteric while five had inferior mesenteric portasystemic shunts. Thus, 7.2 percent of all patients successfully studied by this technique had readily identifiable variceal shunts in proximity to the large bowel.²

Previous abdominal surgery in patients with portal hypertension may be an additional predisposing factor in the development of mesenteric varices at sites of adhesion of bowel to parietal peritoneal structures, such as scars and ostomy sites.³

The role of angiography is firmly established in diagnosing arterial hemorrhage from the lower gastrointestinal tract. Direct extravasation of contrast material from venous lesions such as varices is, however, rarely seen during angiography, even when vasoactive substances such as tolazoline are used.⁴ One must rely on several criteria: exclusion of upper gastrointestinal source by negative results on endoscopy, failure to show arterial bleeding site angiographically during a time of active hemorrhage and visualization of the mesenteric varices with associated hepatofugal flow.³⁻⁵ Transhepatic or transsplenic portography has, however, shown the actual extravasation of contrast material into the bowel.⁶ The practical usage of this technique is limited not only by the lack of wide-

spread availability but also by the prohibitive coagulopathy that makes deep-needle visceral punctures unacceptable in so many patients with cirrhosis.

Colonoscopy, though widely available, is unlikely to provide definitive diagnosis in patients who are bleeding vigorously from mesenteric varices. An unprepared colon filled with stool and clots is an unacceptable endoscopic field. In some patients, such as in the case presented, when hemorrhage ceases and multiple tap-water enemas successfully lavage the colon, limited or even complete colonoscopic examination may be able to confirm the varices while excluding mass lesions, small vascular ectasias and colitis.

Controversy exists over whether primary resection or portacaval shunting is the procedure of choice when patients fail to respond to medical therapy.^{3,4,7} Four of five patients with hemorrhaging colonic varices now seen by one of the authors (M.P.F.) have responded successfully to portal venous decompression.⁴ Although Moncure and associates³ and Fee and co-workers⁷ recommend resection of the varix-bearing bowel with delayed portasystemic shunt, primary portal decompression has several potential advantages. As with esophageal varices, rebleeding from colonic varices has not been a problem following successful portal decompression.⁴ Resection of varix-bearing bowel, however, would be expected to carry with it a high rate of recurrence of bleeding similar to equivalent direct operations on esophageal varices. Also, with primary portal decompression, resection of unprepared bowel with its attendant morbidity is avoided. In our opinion, the advantages of shunt procedure outweigh the potential disadvantages such as encephalopathy and liver failure.

Summary

This is the report of a 35-year-old woman with known cirrhosis and esophageal varices in whom massive hematochezia developed requiring the transfusion of 12 units of blood. The venous phase angiography showed large cecal and ascending colonic varices draining into a right ovarian vein. The patient responded to portacaval shunt. Although infrequently reported as a source of lower gastrointestinal hemorrhage, up to 7 percent of patients with cirrhosis with portal hypertension may well have mesenteric varices concentrated mainly in the cecum. In the unprepared bowel, portal venous surgical decompression may well be preferred to colonic resection. In patients with

portal hypertension, this disease entity should be considered as another potential source of massive lower gastrointestinal hemorrhage.

REFERENCES

1. Edwards EA: Functional anatomy of the portasystemic communications. *Arch Intern Med* 1951 Aug; 88:137-154
2. Nunez D, Russel E, Yrizarry J, et al: Portasystemic communications studied by transhepatic portography. *Radiology* 1978 Apr; 127:75-79
3. Moncure AC, Waltman AC, Vandersaln TJ, et al: Gastrointestinal hemorrhage from adhesion-related mesenteric varices. *Ann Surg* 1976 Jan; 183:24-29
4. Federle M, Clark RA: Mesenteric varices: A source of mesosystemic shunts and gastrointestinal hemorrhage. *Gastrointest Radiol* 1979 Nov; 4:331-337
5. Hamlyn AN, Lunzer MR, Morris JS, et al: Portal hypertension with varices in unusual sites. *Lancet* 1974 Dec; 2:1531-1534
6. Kinkhabwala M, Mousavi A, Iyer S, et al: Bleeding ileal varicosity demonstrated by transhepatic portography. *Am J Roentgenol* 1977 Sep; 129:514-516
7. Fee HJ, Taylor JB, O'Connell TX: Bleeding intestinal varices associated with portal hypertension and previous abdominal surgery. *Am Surg* 1977 Nov; 43:760-762

Refer to: Pont A, Spratt D, Shinn JB: T₃ toxicosis due to non-metastatic follicular carcinoma of the thyroid. *West J Med* 136:255-258, Mar 1982

T₃ Toxicosis Due to Nonmetastatic Follicular Carcinoma of the Thyroid

ALLAN PONT, MD
San Francisco

DANIEL SPRATT, MD
Stanford, California

JOHN B. SHINN, MD
San Jose, California

IT IS UNCOMMON in American medical practice to encounter patients with massive goiters. The cause is rarely diligently researched and is often ascribed to iodine deficiency, inborn error of hormone synthesis or benign multinodular goiter. Therapy is usually offered for cosmetic purposes and includes either levothyroxine sodium or removal by surgery. The possibility that carcinoma constitutes all or part of the goiter may not be considered.

From the Department of Medicine, Endocrinology Division, Santa Clara Valley Medical Center, San Jose, and Stanford University Medical Center (Drs. Pont and Spratt); and the Department of Surgery, Head and Neck Division, Santa Clara Valley Medical Center (Dr. Shinn). Dr. Pont is now Chairman of the Department of Medicine, Children's Hospital of San Francisco.

Submitted, revised, May 18, 1981.

Reprint requests to: Allan Pont, MD, Chairman, Medicine, Children's Hospital of San Francisco, P.O. Box 3805, San Francisco, CA 94119.

ABBREVIATIONS USED IN TEXT

PTU = propylthiouracil
RIA = radioimmunoassay
T₃ = triiodothyronine
T₄ = thyroxine
TSH = thyroid-stimulating hormone

The recent influx of immigrants from Southeast Asia and Mexico includes many patients from "goiter belts." We recently encountered such a patient who had a massive goiter and clinical signs of hyperthyroidism. Laboratory evaluation confirmed triiodothyronine (T₃) toxicosis. The patient was presumed to have a multinodular goiter. Because she had moved from a low-iodine-intake to a high-iodine-intake area, the possibility that the hyperthyroidism was due to the jodbasedow effect was considered, albeit that jodbasedow usually results in a balanced hypersecretion of both thyroxine (T₄) and T₃.¹

Surgical evaluation, however, showed the patient to have a massive follicular carcinoma of the thyroid. No evidence of metastasis was found. The case is presented to document the very rare occurrence of hyperthyroidism due to nonmetastatic follicular carcinoma of the thyroid. The occurrence of thyroid cancer in patients from "goiter" areas and the association of thyroid cancer with hyperthyroidism in general will be reviewed.

Report of a Case

A 52-year-old native-Mexican woman presented in October 1980 desiring cosmetic correction for a massive goiter (Figure 1). She had lived on the desert south of Guadalajara until moving to California six weeks before her clinic visit. She had noticed the swelling in her neck for about four years and felt that there had been some recent growth. She remembered that several other people in her community had had goiters.

The woman said she had not had dysphagia or respiratory problems, but that there had been mild weakness, fatigue, nervousness and heat intolerance. On physical examination the patient was noted to be anxious and moderately obese. Neither lid lag nor exophthalmos was present. Her skin was warm and smooth. There was no proximal muscle weakness, but tremor of the outstretched hands was evident. Blood pressure was 150/75 mm of mercury and pulse was 96 beats per minute. The thyroid gland was firm and